PennAg Industries Association

Serving Agribusiness Since 1878

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August 12, 2004

Division of Dockets Management (HFA-305)
Food and Drug Administration
5630 Fishers Lane
Room 1061
Rockville, MD 20852

Re: Docket No. 2004N0264
(RIN) 0910-AF46

Gentlemen:

My name is Samuel C. Sherk and I am employed by PennAg Industries Association, Harrisburg, PA, where I am the staff liaison to our Feed, Grain and Allied Industries Council. Part of my duties involve keeping our feed manufacturing members aware of state and federal regulations that impact their segment of American agribusiness. I am writing to convey the opinion of our Council in response to the above stated docket number.

In describing BSE and TSE's your background information states that "the agent that causes BSE and TSE's has yet to be fully characterized" and that "the theory most accepted by the scientific community is that the agent is a prion...". You go on to say "the BSE agent is extremely resistant to heat and to normal sterilization processes." We are curious that you refer to a BSE agent in the same paragraph where you admit that you haven't fully characterized the agent that causes BSE and other TSE's. If your initial statement is true the agent that causes TSE's is unknown, therefore, how do you know it's heat resistant? Perhaps an abnormal form of prion protein may be heat resistant but you can't say, based on scientific evidence, that the agent known to cause BSE and other TSE's has been identified.

While we need to be cautious about the introduction of BSE into the United States it is alarming that FDA has launched such a broad sweeping rule based entirely on the theory that an abnormal prion protein may cause BSE and other TSE's. The approach is reminiscent of radioactive cranberries!

Based on our limited understanding of the disease, TSE's occur in mink, cats, cattle, sheep, elk, deer and humans. Why is it that all we hear about are the programs associated with prevention of BSE? We are curious why you are not testing sheep when the disease onset occurs in lambs as early as six months? Could it be that you simply haven't had the congressional pressure to test sheep? How about testing on deer, elk, cats and mink?

We recall that cases of CWD in wild deer led to mass hunting and herd reductions of deer in several Wisconsin counties. Wisconsin is the nation's leading producer of mink pelts. Is there any possibility that protein from mink carcasses somehow became available as a protein source for wild deer in Wisconsin?

We fully understand the hysteria that a single case of CJD or vCJD in a US citizen may create, despite the fact that 150 known cases of vCJD in a world population of 8 billion is hardly an epidemic. Unfortunately, the reaction to those cases, which have occurred, primarily in Europe, has caused a furor and subsequent rulings based on the premise that those people affected by vCJD may have eaten beef while in Europe. We are curious about the number of these people who ate lamb or mutton while in Europe. Do they test sheep in the UK?

The rules already in place may not be responsible for the prevention of anything. You cannot claim victory in this process without knowing the exact cause of TSE's and how these rules contributed to their prevention.

We are appalled at the gross overkill suggested by the proposed rule, in the docket noted above, and hope you give serious consideration to the sweeping economic changes the rule will create. Specifically, there is no valid scientific reason to proceed beyond the safeguards already in place. We urge you to exercise caution in the adoption of rulings, based solely on theory, that go too far, too fast.

Sincerely,

Samuel C. Sherk
Assistant Vice President

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